

tion of inflammation after repair in IAAAs. This skepticism is unfounded based on our results in which inflammation improved in all cases of EVAR.

Numerous reports demonstrate, similar to our present series, that EVAR treatment for inflammatory aneurysms successfully reduces the size of the aortic aneurysm in a majority of patients. It also reduces the amount of PAF but not to the extent that open aneurysm repair does. Endovascular repair outcomes are achieved with a lower morbidity and 1-year mortality. The Achilles' heel of EVAR repair for IAAAs revolves around renal and ureter involvement. Hydroureter and hydronephrosis demonstrate inferior outcomes with EVAR compared to open repair. This may be related to the fact that the PAF resolves at a slower and less robust rate. However, this is purely a hypothesis. Based on our results and results in the literature, one would suggest that EVAR is appropriate treatment for IAAAs where there is no renal or ureter involvement in the inflammation. It may also be successful treatment for a percentage of patients in which they are poor open surgical risks.

AUTHOR CONTRIBUTIONS

Conception and design: WS, GF, SM, TB

Analysis and interpretation: WS, GF, SM, TB, GO

Data collection: WS, GF, GO, MK, SN

Writing the article: WS, SM

Critical revision of the article: WS, GF, TB, GO, MK, SN, SM

Final approval of the article: WS, GF, TB, GO, MK, SN, SM

Statistical analysis: WS

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Overall responsibility: WS

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DISCUSSION

Dr Thomas Naslund, MD (*Nashville, Tenn*). The authors have succeeded in advancing our knowledge of the role EVAR holds in treating this uncommon but complex vascular disease. Results from open as well as endovascular repair in this series are admirable and represent a standard all of us can look up to in our own efforts to manage these problematic aneurysms.

Your series is large for a study involving IAAAs, only 10 patients were treated with EVAR, whereas 59 were treated by open repair. Your proportion of patients managed with EVAR is similar to other practices favoring open repair. One reason that IAAAs are infrequently managed with EVAR is the opinion that reduction in inflammatory change is suboptimal. This report demonstrates that inflammatory findings on CT scan improve after EVAR and, based on correlations with other studies, likely similar to the reduction of inflammation seen in the aortic sac after open repair. However, your results support the premise that resolution of retroperitoneal

fibrosis and ureteral entrapment is less complete with EVAR than with open repair. This finding should be considered when indication for surgery is ureteral entrapment.

I pose three questions to the authors. (1) What happens to the patients' pain from IAAA after EVAR? (2) Should steroids be added to EVAR to improve resolution of inflammation? (3) Have you identified any technical tips in performing EVAR in IAAA?

Dr Grant T. Fankhauser. Thank you, Dr Naslund, I appreciate your kind comments and I appreciate your questions. First of all, in regard to the pain scenario, it is very difficult in the retrospective review to have truly analyzed the pain and whether the pain was resolved after the procedure. That really was not part of our review. I will say though that the majority of the patients who had smaller aneurysms underwent repair of these aneurysms because they did have symptoms, most commonly, pain. So, I do not have a direct answer to what the relation of that is. When you look at the literature, there is very little information that will relate to

pain and resolution of pain after the procedure. In the question regarding the addition of steroids, in the United Kingdom meta-analysis, there is no question that addition of steroids in those small select patients who had hydronephrosis was beneficial. It did add to the resolution of the hydronephrosis. In our small series, with only a few people who actually had steroid therapy, it is difficult for me to make any meaningful statements about the effects of steroid therapy, but there is literature that would suggest that adding steroids in those patients who had recalcitrant hydronephrosis is a reasonable option. Tips regarding endovascular repair when you are preparing an inflammatory aneurysm vs an atherosclerotic

aneurysm, because of the issue with limb kinkage and limb stenosis, I think you need to be very careful when you are doing your postimaging to be sure that there is not a kink or a significant stenosis. You may find yourself using some sort of self-expanding stent or another stent on the inside to try to prevent that or try to keep that open. Because of the fibrotic change on the outside, it may cause more of a stenosis and, because you do not know for sure whether that fibrosis is going to resolve, then I would suggest that you make sure that they are widely patent when you are done. Other than that, you repair them pretty much in the same manner as you would an atherosclerotic aneurysm.

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